THE SEAT OF ABSINTHIC EPILEPSY.[1]

By ISAAC OTT, M.D.

Easton, Pa.

THE seat of epilepsy has not been revealed either by coarse or fine anatomy. Much more has been accomplished by physiology in the elucidation of convulsive phenomena. Thus Brown-Sequard found that section of the sciatic, or injuries of the medulla, cerebral peduncle, corpora quadrigemina and spinal cord were often sufficient to produce epilepsy. His epileptogenic zone at the angle of the jaw on the side of the lesion is a favorite means to produce epilepsy at will. Further, the young of animals rendered epileptic by his proceedures were often spontaneously epileptic.

The anæmic theory of epilepsy at one time held a large sway, due to the experiments of Küssmaul and Tenner, who produced epileptiform convulsions by creating an artificial anæmia of the brain. The pallid face of epileptics has been supposed to support this theory, but neither flushing nor paleness of the skin is any proof of the state of the circulation in the profundity of the body. Local hyperæmias and anæmias of the skin are quite common, and are only skin deep. Kürsmaul and Tenner found that somewhere between the spinal cord and crura cerebri was a convulsive point; Nothnagel located what he calls a convulsive centre in the pons variolii; Ziehen found that irritation of the posterior part of the corpora quadrigemina caused convulsions. But Hughlings-Jackson has put forth the theory that these convulsions are due to explosions of nerve force in the gray matter of the cerebrum. Horsely found that irritating the cortex generated tonic as well as clonic impulses.

Magnan in experimenting with absinthe arrived at the conclusion that the drug excites simultaneously the spinal cord and the cerebral centres; but Horsely has shown that convulsions do not appear if the cord is cut at the eighth dorsal vertebra. He has also discovered that if the motor centres are removed, convulsions do not take place on the opposite side of lesion, excepting a slight tonus. According to this observer, after the injection *per jugular* of two drops of essence of absinthe there follows the following phenomena: the facial muscles begin with single clonic spasms, passing into a state of tremulous tonic spasm. This order of convulsions passes rapidly down the body until the tonic spasm in the limbs is extremely marked. After a short period tonic spasm gives way to a long series of clonic twitches. Accompanying these convulsive phenomena there is a profuse salivation, and sometimes escape of urine, while in cases in which narcosis has not been employed, unconsciousness and coma are early symptoms. He also tested the effect of absinthe upon the cord by means of the electrical method, using both the galvanometer and electrometer. His results showed that the employment of absinthe gave maximal electro-motive effects in the sciatic nerve through the overwhelming discharge of the highest cortical centres. He also observed that absinthe does not exhaust the cortex, for after stimulation of the absinthe an additional electric excitation caused the centres to produce more energy.

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Now absinthe not only produces a typical epilepsy in animals, but it also causes it in man. It is evident that in this medicament we have a most valuable means to study the origin and seat of the convulsive disorder. That the convulsions are not due to circulatory changes is well seen in experiments which show no change of pressure before the commencement of a fit, but during the fit a rise of tension, a fact already announced by Todorsky and Bechterew. These observers as well as Horsley have found the brain hyperæmic, rather than anæmic. All these facts go to show that these convulsions are not due to circulatory changes. There remains to determine their mode of origin in the nervous system. Magnan believed their source to be spinal, but Horsely contradicts this; and of the thirty-five experiments made with absinthe I have seen no spinal convulsions. If the whole cortex is removed in an animal, then tonic convulsions ensue not usually succeeded by clonic acts. If the corpora striata or optic thalami are divided still spasms of a tonic nature continue with no clonic stage. The same results ensue after removal of the corpora quadrigemina. If, however, the pons is divided, then all convulsive acts cease.

If in an animal the motor centres are removed on one side, then tonic spasms ensued on the side opposite the lesion, whilst tonic and clonic convulsions took place on the side of the lesion. The convulsive movements upon the side opposite the lesion are weaker than when the cortex is intact. Irritation of the cortex in the hands of other observers has shown that it calls out tonus and clonus. Further, if the cortex is cut off and we then excite the underlying fibres we only obtain tonus, a fact observed by two French observers, Franck and Pitres, and confirmed by others. If a fit is in full progress Munk has observed that instantaneous slicing off of the cortical gray immediately arrested the fit. All these facts go to show that the seat of origin of tonic and clonic movements in epilepsy is in the cortex, and their expression takes place mainly by means of ganglia seated in the pons varolii.

That no clonic convulsions can be observed after removal of the brain and part of the pons is not always true, for I have seen violent clonic spasms ensue in one case upon the injection of essence of absinthe. Electrical irritation of the pons can give clonic convulsions, as has already been noted by Todd.

The word cortico-frontal might express the origin of convulsive epilepsy due to absinthe.

Of the thirty-five experiments upon rabbits, the above notes will suffice for their explanation without going into detailed description.